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Recommendations of the Immunization Practices Advisory Committee

Prevention of Perinatal Transmission of Hepatitis B Virus: Prenatal Screening of all Pregnant Women for Hepatitis B Surface Antigen

Transmission of hepatitis B virus (HBV) from mother to infant during the perinatal period represents one of the most efficient modes of HBV infection and often leads to severe long-term sequelae. Infants born to mothers positive for hepatitis B surface antigen (HBsAg) and hepatitis B "e" antigen (HBeAg) have a 70%–90% chance of acquiring perinatal HBV infection, and 85%–90% of infected infants will become chronic HBV carriers (*1,2*). It has been estimated that more than 25% of these carriers will die from primary hepatocellular carcinoma or cirrhosis of the liver (*3*). These deaths usually occur during adulthood, when familial and financial responsibilities make them particularly devastating. In the United States, an estimated 16,500 births occur to HBsAg-positive women each year (about 4,300 of whom are also HBeAg-positive), and approximately 3,500 of these infants become chronic HBV carriers. Prenatal screening of all pregnant women would identify those who are HBsAg-positive and thus would allow treatment of their newborns with hepatitis B immune globulin (HBIG) and hepatitis B (HB) vaccine, a regimen that is 85%–95% effective in preventing the development of the HBV chronic carrier state (*2,4–6*).

In 1984, the Immunization Practices Advisory Committee (ACIP) recommended that pregnant women in certain groups at high risk for HBV infection be screened for HBsAg during a prenatal visit and, if found to be HBsAg-positive, that their newborns receive HBIG and HB vaccine at birth (*7*). No data are available regarding the proportion of high-risk women currently being screened in clinical practice, but several studies and the experience of public health workers indicate that major problems have been encountered in implementing these recommendations (*8–12*). These include 1) concerns about the sensitivity, specificity, and practicality of the current ACIP guidelines for identifying HBV carrier mothers; 2) lack of knowledge among prenatal health-care providers about the risks of perinatal transmission of HBV and about recommended screening and treatment procedures; 3) poor coordination among medical-care workers who provide treatment and follow-up of mothers and infants; and 4) refusal of some public and private third-party payers to reimburse for HBV screening of pregnant women and treatment of their infants. In addition,

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concern has been expressed that these recommendations may not be practical or applicable in some U.S. jurisdictions where HBV infection is highly endemic, such as parts of Alaska and certain Pacific Islands.

The problems encountered in implementing the currently recommended strategy of screening high-risk women have been examined by a number of investigators. Recent studies in several large inner-city hospitals, where all pregnant women were tested for HBsAg, have found that only about 35%–65% of HBsAg-positive mothers would have been identified by following the current ACIP guidelines (*8–12*). In these studies, the prevalence of HBsAg in inner-city black (0.4%–1.5%) and Hispanic women was higher than expected. Several investigators expressed concern that many health-care providers are too busy or may be reluctant to obtain the sexual and drug-use history necessary to identify high-risk patients for screening. In addition, persons providing health care to pregnant women often are not aware of the risks of perinatal transmission of HBV and of the recommended screening and treatment guidelines. In one study, 40% of obstetricians could name no more than two groups at high risk for HBV infection, and only 28% knew the recommended treatment for infants born to HBV carrier mothers (CDC, unpublished data).

Given these limitations, it is now evident that routine screening of all pregnant women is the only strategy that will provide acceptable control of perinatal transmission of HBV infection in the United States. Screening the approximately 3.5 million pregnant women per year for HBsAg would identify 16,500 positive women and allow treatment that would prevent about 3,500 infants from becoming HBV carriers. Recent studies also indicate that the costs and benefits of universal testing of mothers are comparable to those encountered in other widely implemented programs of prenatal and blood-donor screening (*13,14*). The cost of an HBsAg test ranges from an estimated $3.50 per test in blood-bank laboratories to $21.00 per test in private commercial laboratories. If one assumes an average screening cost ranging from $12.00 to $20.00 per test plus $150.00 for the HBIG and vaccine needed to treat each infant of an HBsAg-positive mother, the cost to prevent one newborn infant from becoming a chronic HBV carrier would be between $12,700 and $20,700.

HBsAg testing should be done early in pregnancy when other routine prenatal testing is done. The HBsAg test is widely available and can be added to the routine prenatal "panel" of tests without requiring additional patient visits. The advantages of making HBsAg testing routine during early pregnancy include 1) the ability to identify HBV carrier mothers that is not dependent on the health-care provider's identifying high-risk women or ordering HBsAg as a special test; 2) the availability of test results before delivery so that infants can receive HBIG and vaccine without delay after birth; and 3) appropriate counseling of families before delivery (*15*).

Because more than 90% of women found to be HBsAg-positive on routine screening will be HBV carriers, routine follow-up testing later in pregnancy is not necessary for the purpose of screening. In special situations, such as when the mother is thought to have acute hepatitis, when there has been a history of exposure to hepatitis, or when particularly high-risk behavior such as parenteral drug abuse has occurred during the pregnancy, an additional HBsAg test can be ordered during the third trimester. Few women in populations at low risk for HBV infection will have a change in HBsAg status during subsequent pregnancies. However, because of the expected benefits of making HBsAg testing a routine part of each prenatal panel, testing should be done during each pregnancy.

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Women who present for delivery without prenatal care or without medical records documenting the results of HBsAg screening should have the HBsAg test done as soon as possible after admission, since delay in administration of HBIG to infants of carrier mothers will decrease the efficacy of therapy. In the studies that demonstrated the highest efficacy (85%–95%) of combined HBIG and HB vaccine prophylaxis, HBIG was administered within 2–12 hours after birth (*2,4–6*). In one study in which only HBIG was used for prophylaxis, no efficacy was found if HBIG was given more than 7 days after birth, and a significant decrease in efficacy was observed if it was given more than 48 hours after birth (*16*). Only one-third of U.S. hospitals currently perform the HBsAg test as an in-house procedure, and many of these have technicians who are trained to do the test available on only one shift. Hospitals that cannot rapidly test for HBsAg should either develop this capability or arrange for testing to be done at a local laboratory or blood bank where test results can be obtained within 24 hours.

The commercially available HBsAg tests have an extremely high sensitivity and specificity if positive tests are repeated and confirmed by neutralization as recommended by the manufacturers of the reagent kits. Testing for other markers of HBV infection, such as HBeAg, is not necessary for maternal screening. Mothers who are positive for both HBsAg and HBeAg have the highest likelihood of transmitting HBV to their newborns. However, infants of mothers who are HBsAg-positive but HBeAg-negative may become infected and develop severe, even fatal, fulminant hepatitis B during infancy (*17,18*). For this reason, HBIG and HB vaccine treatment of all babies born to HBsAg-positive women is recommended.

HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by a physician. Identification of women who are HBV carriers through prenatal screening presents an opportunity to vaccinate susceptible household members and sexual partners of HBV carriers, as previously recommended (*19*). Screening and vaccination of susceptible contacts should be done by the family's pediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant women.

Implementation of the recommendations to prevent perinatal transmission requires maternal screening, treatment of the newborn in the hospital, and administration of subsequent doses of HB vaccine to the infant during pediatric visits at 1 and 6 months of age. This multistep process requires effective transfer of information among several groups of health-care providers, knowledge of recommended treatment, and availability of HBIG and vaccine at separate facilities. Treatment failures due to lack of communication among health-care providers can occur, especially in situations where prenatal, obstetric, and pediatric care are provided in different facilities (*20*). Central coordination of the treatment of these infants by city, county, or state health departments would improve the education of the health-care providers involved and increase the likelihood that proper treatment is provided.

In certain populations under U.S. jurisdiction, including Alaskan Natives and Pacific Islanders, as well as in many other parts of the world, HBV infection is highly endemic in the general population, and transmission occurs primarily during childhood (*21*). In such groups, universal vaccination of newborns with HB vaccine is recommended to prevent disease transmission both during the perinatal period and during childhood. Several studies have shown that HB vaccine given without HBIG will prevent 70%–85% of perinatal HBV infections and 95% of early childhood infections (*22,23*). In many of these areas with highly endemic HBV infection, prenatal

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screening is impractical because the population is isolated, laboratory facilities are not available, and/or health-care budgets and personnel are limited. In these areas, control of HBV infection can be better achieved by directing available resources into programs to vaccinate all children with HB vaccine. Programs for screening all mothers for HBsAg and providing HBIG to infants born to carrier mothers are costly and will add only modestly to disease prevention. They should be considered only after the program for universal vaccination of children has been implemented.

RECOMMENDATIONS

All pregnant women should be routinely tested for HBsAg during an early prenatal visit in each pregnancy. This testing should be done at the same time that other routine prenatal screening tests are ordered. In special situations, such as when acute hepatitis is suspected, when there has been a history of exposure to hepatitis, or when the mother has a particularly high-risk behavior such as intravenous drug abuse, an additional HBsAg test can be ordered later in the pregnancy.

If a woman has not been screened prenatally or if test results are not available at the time of admission for delivery, HBsAg testing should be done at the time of admission, or as soon as possible thereafter. If the mother is identified as HBsAg-positive more than 1 month after giving birth, the infant should first be tested for HBsAg; if negative, the infant should be treated with HBIG and HB vaccine. Hospitals where infants are delivered should have HBsAg testing capabilities or should be able to obtain HBsAg results within 24 hours from a local laboratory.

If a serum specimen is positive for HBsAg, the same specimen should be tested again, and then the test results should be confirmed by neutralization. It is unnecessary to test for other HBV markers during maternal screening, although HBsAg-positive mothers identified during screening may have HBV-related acute or chronic liver disease and should be evaluated by their physician.

Infants born to HBsAg-positive mothers should receive HBIG (0.5 mL) intramuscularly (IM) once they are physiologically stable, preferably within 12 hours after birth. HB vaccine, either plasma-derived (10 μg per dose) or recombinant (5 μg per dose), should be administered IM in three doses of 0.5 mL each. The first dose should be given concurrently with HBIG but at a different site. If vaccine is not immediately available, the first dose can be given within 7 days after birth. The second and third doses should be given 1 month and 6 months after the first. Testing the infant for HBsAg and its antibody (anti-HBs) is recommended at 12–15 months of age to monitor the effectiveness of therapy. If HBsAg is not detectable and anti-HBs is present, the child can be considered protected. Testing for antibody to hepatitis B core antigen (anti-HBc) is not useful, since maternal anti-HBc can persist for more than a year. HBIG and HB vaccination do not interfere with the routine childhood immunizations.

Household members and sexual partners of HBV carriers identified through prenatal screening should be tested to determine susceptibility to HBV infection and, if susceptible, should receive HB vaccine. Screening and vaccination of susceptible contacts should be done by the family's pediatrician, primary health-care provider, or the physician evaluating the clinical status of the HBsAg-positive pregnant women.

Obstetric and pediatric staff should be notified directly about HBsAg-positive mothers so that the neonate can receive therapy without delay after birth and follow-up doses of vaccine can be given. Hospitals, as well as state, county, and city health departments, should establish programs to educate appropriate health-care

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providers about perinatal transmission of HBV and its control through maternal screening, treatment of infants, and vaccination of susceptible household and sexual contacts of HBV carrier women.

Programs to coordinate the activities of those providing prenatal care, hospital-based obstetrical services, and pediatric well-baby care must be established to assure proper follow-up and treatment of infants born to HBsAg-positive mothers and other susceptible household and sexual contacts.

In populations under U.S. jurisdiction in which hepatitis B infection is highly endemic, including certain Alaskan Native and Pacific Island groups, vaccination of all newborns with HB vaccine is the most effective strategy for HB control. In these populations, such vaccination programs should be given highest priority. In areas where HBsAg screening of mothers and use of HBIG in infants born to HBV carrier mothers are not practical, the vaccination of all newborns with HB vaccine should be considered the appropriate treatment.

Editorial Note: Hepatitis B vaccine is the first human vaccine that can prevent both serious chronic disease and a uniformly fatal type of cancer. These recommendations, developed in consultation with representatives of the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics, represent a major step toward control of perinatal hepatitis B transmission in the United States. Programs for universal screening of pregnant women are currently in progress in Hawaii, certain Canadian provinces, Italy, West Germany, New Zealand, Australia, and Japan. More extensive infant HB vaccination programs are in progress in Alaska, American Samoa, Korea, Taiwan, Singapore, and the People's Republic of China. A number of U.S. health-care facilities have already begun to screen all pregnant women for HBsAg.

State and local health departments can facilitate implementation of these recommendations by 1) working to assure that all women receiving prenatal care in both public and private sector programs are offered screening and appropriate treatment; 2) working to assure that costs of screening and treatment are covered by public and private third-party payers; 3) establishing programs to coordinate the transfer of information between prenatal, obstetric, and pediatric health-care providers; and 4) providing health education about hepatitis B to the public and to health-care providers. CDC will continue to work with state and local health agencies and professional associations in hepatitis B prevention and control.

References

1. Stevens CE, Beasley RP, Tsui J, Lee W-C. Vertical transmission of hepatitis B antigen in Taiwan. N Engl J Med 1975;292:771–4.
2. Stevens CE, Toy PT, Tong MJ, et al. Perinatal hepatitis B virus transmission in the United States: prevention by passive-active immunization. JAMA 1985;253:1740–5.
3. Beasley RP, Hwang L-Y. Epidemiology of hepatocellular carcinoma. In: Vyas GN, Dienstag JL, Hoofnagle JH, eds. Viral hepatitis and liver disease. Orlando, Florida: Grune & Stratton, 1984:209–24.
4. Beasley RP, Hwang L-Y, Lee GC-Y, et al. Prevention of perinatally transmitted hepatitis B virus infections with hepatitis B immune globulin and hepatitis B vaccine. Lancet 1983;2:1099–102.
5. Wong VCW, Ip HMH, Reesink HW, et al. Prevention of the HBsAg carrier state in newborn infants of mothers who are chronic carriers of HBsAg and HBeAg by administration of hepatitis-B vaccine and hepatitis-B immunoglobulin: double-blind randomised placebo-controlled study. Lancet 1984;1:921–6.

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6. Stevens CE, Taylor PE, Tong MJ, et al. Yeast-recombinant hepatitis B vaccine: efficacy with hepatitis B immune globulin in prevention of perinatal hepatitis B virus transmission. JAMA 1987;257:2612–6.
7. Immunization Practices Advisory Committee. Postexposure prophylaxis of hepatitis B. MMWR 1984;33:285–90.
8. Kumar ML, Dawson NV, McCullough AJ, et al. Should all pregnant women be screened for hepatitis B? Ann Intern Med 1987;107:273–7.
9. Jonas MM, Schiff ER, O'Sullivan MJ, et al. Failure of Centers for Disease Control criteria to identify hepatitis B infection in a large municipal obstetrical population. Ann Intern Med 1987;107:335–7.
10. Summers PR, Biswas MK, Pastorek JG II, Pernoll ML, Smith LG, Bean BE. The pregnant hepatitis B carrier: evidence favoring comprehensive antepartum screening. Obstet Gynecol 1987;69:701–4.
11. Wetzel AM, Kirz DS. Routine hepatitis screening in adolescent pregnancies: is it cost effective? Am J Obstet Gynecol 1987;156:166–9.
12. Delage G, Montplaisir S, Rémy-Prince S, Pierri E. Hepatitis B Virus Immunization Study Group. Prevalence of hepatitis B virus infection in pregnant women in the Montreal area. Can Med Assoc J 1986;134:897–901.
13. Arevalo JA, Washington AE. Cost-effectiveness of prenatal screening and immunization for hepatitis B virus. JAMA 1988;259:365–9.

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TABLE I. Summary — cases of specified notifiable diseases, United States

Disease	22nd Week Ending			Cumulative, 22nd Week Ending		
	Jun. 4, 1988	Jun. 6, 1987	Median 1983-1987	Jun. 4, 1988	Jun. 6, 1987	Median 1983-1987
Acquired Immunodeficiency Syndrome (AIDS)	740	U*	122	13,328	7,539	2,925
Aseptic meningitis	76	143	102	1,648	2,056	1,788
Encephalitis: Primary (arthropod-borne & unspec)	14	20	17	276	366	366
Post-infectious	2	6	3	42	44	47
Gonorrhea: Civilian	11,477	14,477	13,867	278,486	333,235	348,262
Military	199	299	270	5,081	7,122	8,588
Hepatitis: Type A	366	479	330	9,921	10,501	9,293
Type B	359	518	490	8,791	10,663	10,441
Non A, Non B	45	67	73	1,033	1,327	1,475
Unspecified	30	43	83	889	1,357	2,060
Legionellosis	15	18	15	316	362	266
Leprosy	-	6	5	72	90	111
Malaria	12	11	18	268	314	314
Measles: Total†	74	114	68	1,439	2,216	1,474
Indigenous	73	109	62	1,307	1,952	1,327
Imported	1	5	6	132	264	169
Meningococcal infections	32	42	42	1,483	1,542	1,480
Mumps	168	328	66	2,556	6,545	1,840
Pertussis	42	31	31	897	728	752
Rubella (German measles)	5	3	14	96	174	249
Syphilis (Primary & Secondary): Civilian	647	753	460	15,850	14,217	11,809
Military	5	4	3	82	76	89
Toxic Shock syndrome	2	3	7	121	131	170
Tuberculosis	405	487	370	8,101	8,507	8,507
Tularemia	4	4	5	50	51	55
Typhoid Fever	4	3	4	142	126	127
Typhus fever, tick-borne (RMSF)	13	27	32	82	90	127
Rabies, animal	91	97	110	1,682	2,167	2,174

TABLE II. Notifiable diseases of low frequency, United States

	Cum. 1988		Cum. 1988
Anthrax	-	Leptospirosis	12
Botulism: Foodborne (Md. 1)	8	Plague	1
Infant (Tex. 1)	16	Poliomyelitis, Paralytic	-
Other	2	Psittacosis (Pa. 1; Colo. 2)	33
Brucellosis (Tex. 1; Calif. 1)	24	Rabies, human	-
Cholera	-	Tetanus	18
Congenital rubella syndrome	3	Trichinosis	8
Congenital syphilis, ages < 1 year	-		
Diphtheria	-		

*Because AIDS cases are not received weekly from all reporting areas, comparison of weekly figures may be misleading.
†There were no cases of internationally imported measles reported for this week.

TABLE III. Cases of specified notifiable diseases, United States, weeks ending June 4, 1988 and June 6, 1987 (22nd Week)

Reporting Area	AIDS	Aseptic Meningitis	Encephalitis		Gonorrhea (Civilian)		Hepatitis (Viral), by type				Legionellosis	Leprosy
			Primary	Post-infectious			A	B	NA,NB	Unspecified		
	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	13,328	1,648	276	42	278,488	333,235	9,921	8,791	1,033	889	316	72
NEW ENGLAND	517	71	10	-	8,416	10,888	356	520	75	48	18	10
Maine	16	5	1	-	187	325	14	23	3	1	2	-
N.H.	15	10	-	-	117	184	26	32	4	3	1	-
Vt.	4	4	3	-	65	85	4	15	5	-	1	-
Mass.	264	30	5	-	3,006	4,007	179	331	51	39	12	9
R.I.	29	18	-	-	777	862	46	55	8	-	2	1
Conn.	190	4	1	-	4,262	5,425	87	64	4	5	-	-
MID. ATLANTIC	4,572	176	32	1	42,259	52,897	604	1,154	67	93	67	8
Upstate N.Y.	679	99	20	1	5,610	6,832	361	321	34	10	35	-
N.Y. City	2,490	30	7	-	17,825	28,158	120	528	7	63	11	5
N.J.	991	47	5	-	6,515	6,764	111	287	23	20	-	1
Pa.	412	-	-	-	12,309	11,143	12	18	3	-	21	-
E.N. CENTRAL	992	222	64	5	43,880	48,038	518	992	54	46	76	-
Ohio	221	81	22	2	10,625	10,372	152	238	16	8	29	-
Ind.	78	29	9	-	3,276	4,019	60	140	7	16	5	-
Ill.	458	35	12	3	12,516	14,444	69	85	-	4	-	-
Mich.	194	69	16	-	14,293	14,815	164	329	21	18	32	-
Wis.	41	8	5	-	3,170	4,388	73	100	10	-	10	-
W.N. CENTRAL	274	76	18	4	11,079	13,398	617	435	47	16	33	-
Minn.	52	16	2	1	1,511	2,097	35	63	6	3	1	-
Iowa	14	15	8	-	823	1,296	30	43	8	-	9	-
Mo.	149	22	1	-	6,232	6,787	350	256	22	8	5	-
N. Dak.	1	-	-	-	70	133	2	3	1	3	1	-
S. Dak.	4	6	-	1	210	260	1	2	2	-	11	-
Nebr.	16	3	2	2	661	806	18	19	-	-	4	-
Kans.	38	14	5	-	1,572	2,009	181	49	8	2	2	-
S. ATLANTIC	2,212	384	37	16	80,176	87,392	874	1,834	151	137	65	1
Del.	18	9	2	-	1,141	1,274	15	52	5	1	6	-
Md.	254	41	4	3	8,201	9,673	119	293	13	6	9	1
D.C.	206	8	-	1	5,626	5,961	9	22	3	1	-	-
Va.	146	45	15	2	5,434	6,575	173	119	34	93	6	-
W. Va.	6	7	1	-	579	686	7	28	2	3	-	-
N.C.	128	66	11	-	12,777	13,287	161	334	32	-	22	-
S.C.	74	5	-	1	5,815	7,104	26	248	6	3	10	-
Ga.	314	40	1	-	15,887	14,956	156	274	7	3	6	-
Fla.	1,066	164	3	9	24,916	27,876	208	484	49	27	6	-
E.S. CENTRAL	361	112	22	5	21,568	24,469	367	550	71	6	9	1
Ky.	43	36	6	1	2,036	2,497	320	100	30	2	4	-
Tenn.	177	11	6	-	7,134	8,476	28	277	19	-	2	-
Ala.	88	53	10	2	7,123	7,856	7	139	17	4	2	1
Miss.	53	13	-	2	5,275	5,640	12	34	5	-	1	-
W.S. CENTRAL	1,117	171	20	-	31,648	37,825	1,007	664	81	216	9	13
Ark.	42	3	2	-	2,869	3,784	123	38	1	4	2	-
La.	173	31	3	-	6,632	6,880	61	157	13	9	3	-
Okla.	68	15	4	-	2,860	4,152	220	71	20	17	4	-
Tex.	834	122	11	-	19,287	23,009	603	398	47	186	-	13
MOUNTAIN	398	72	19	1	6,036	8,882	1,462	714	116	89	16	-
Mont.	8	2	-	-	194	215	21	26	6	3	-	-
Idaho	4	1	-	-	175	314	63	46	3	1	-	-
Wyo.	3	1	-	-	100	172	1	5	3	-	1	-
Colo.	149	24	3	-	1,370	1,877	99	93	26	42	5	-
N. Mex.	22	4	2	-	562	940	258	101	7	1	-	-
Ariz.	129	21	5	-	2,154	3,142	741	280	40	25	7	-
Utah	33	11	4	1	249	278	169	65	23	13	2	-
Nev.	50	8	5	-	1,232	1,944	101	98	8	4	1	-
PACIFIC	2,885	364	54	10	33,426	49,446	4,126	2,028	371	238	23	41
Wash.	175	-	3	4	2,522	3,652	938	292	67	21	6	2
Oreg.	83	-	-	-	1,279	1,853	672	260	37	11	-	1
Calif.	2,570	319	48	6	28,878	42,765	2,383	1,424	262	199	14	34
Alaska	10	8	2	-	450	761	127	29	4	4	-	1
Hawaii	47	37	1	-	297	415	6	23	1	3	3	3
Guam	1	-	-	-	56	87	3	3	-	2	1	3
P.R.	626	14	2	-	621	952	15	112	20	20	-	-
V.I.	9	-	-	-	170	110	1	3	2	-	-	-
Amer. Samoa	-	-	-	-	23	39	-	1	-	-	-	-
C.N.M.I.	-	-	-	-	19	-	1	2	-	4	-	-

N: Not notifiable U: Unavailable C.N.M.I.: Commonwealth of the Northern Mariana Islands

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending June 4, 1988 and June 6, 1987 (22nd Week)

Reporting Area	Malaria	Measles (Rubeola)					Meningococcal Infections	Mumps		Pertussis			Rubella		
		Indigenous		Imported*		Total									
	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	Cum. 1988	1988	Cum. 1988	1988	Cum. 1988	Cum. 1987	1988	Cum. 1988	Cum. 1987
UNITED STATES	288	73	1,307	1	132	2,216	1,493	168	2,556	42	897	728	5	96	174
NEW ENGLAND	24	-	19	-	46	191	119	50	87	1	79	18	-	1	1
Maine	2	-	-	-	-	3	3	-	-	-	11	1	-	-	1
N.H.	-	-	13	-	44	149	14	50	83	-	22	2	-	-	-
Vt.	-	-	-	-	-	18	6	-	1	-	2	3	-	-	-
Mass.	16	-	1	-	-	5	50	-	3	1	34	4	-	-	-
R.I.	4	-	-	-	-	1	19	-	-	-	1	-	-	1	-
Conn.	2	-	5	-	2	15	27	-	-	-	9	8	-	-	-
MID. ATLANTIC	33	33	528	-	23	420	144	5	213	-	36	105	-	9	7
Upstate N.Y.	18	-	4	-	2	23	70	1	43	-	21	80	-	1	5
N.Y. City	10	1	25	-	1	351	28	4	79	-	1	-	-	5	1
N.J.	5	-	2	-	11	9	45	-	25	-	4	5	-	1	1
Pa.	2	32	497	-	9	37	1	-	66	-	10	20	-	1	-
E.N. CENTRAL	14	20	96	1	18	269	163	4	532	1	102	89	-	21	21
Ohio	2	-	2	-	4	5	66	-	68	-	21	26	-	-	-
Ind.	-	11	30	-	-	-	18	1	43	1	51	1	-	-	-
Ill.	-	9	51	15	10	101	6	3	196	-	2	5	-	17	19
Mich.	11	-	13	-	4	27	50	-	161	-	18	26	-	4	2
Wis.	1	-	-	-	-	136	23	-	74	-	10	31	-	-	-
W.N. CENTRAL	8	-	10	-	-	135	58	2	111	-	38	39	-	-	1
Minn.	4	-	10	-	-	23	14	-	-	-	7	8	-	-	-
Iowa	-	-	-	-	-	-	-	1	30	-	14	6	-	-	1
Mo.	3	-	-	-	-	110	21	1	28	-	6	13	-	-	-
N. Dak.	-	-	-	-	-	1	-	-	-	-	6	2	-	-	-
S. Dak.	-	-	-	-	-	-	2	-	-	-	2	2	-	-	-
Nebr.	-	-	-	-	-	-	6	-	11	-	-	-	-	-	-
Kans.	1	-	-	-	-	1	15	-	42	-	3	8	-	-	-
S. ATLANTIC	39	15	241	-	11	69	268	31	359	6	89	146	-	3	12
Del.	-	-	-	-	-	18	1	-	-	-	3	-	-	-	2
Md.	3	-	5	-	2	-	24	12	66	-	17	4	-	-	2
D.C.	5	-	-	-	-	1	7	1	119	-	-	-	-	-	-
Va.	8	15	144	-	2	-	30	14	95	5	12	34	-	-	1
W. Va.	-	-	6	-	-	-	2	-	6	-	-	22	-	-	-
N.C.	9	-	-	-	1	2	48	3	31	1	26	61	-	-	-
S.C.	3	-	-	-	-	-	30	-	4	-	-	-	-	-	-
Ga.	3	-	-	-	-	-	41	-	19	-	17	17	-	-	1
Fla.	8	-	86	-	6	48	85	1	19	-	14	8	-	3	6
E.S. CENTRAL	5	1	43	-	-	2	147	11	335	1	14	11	-	-	2
Ky.	-	-	32	-	-	-	29	6	146	-	-	1	-	-	2
Tenn.	-	-	-	-	-	-	90	5	180	-	8	3	-	-	-
Ala.	4	-	-	-	-	-	19	-	6	1	5	5	-	-	-
Miss.	1	1	11	-	-	2	9	N	N	-	1	2	-	-	-
W.S. CENTRAL	27	-	9	-	-	178	95	45	498	2	65	43	-	7	5
Ark.	-	-	-	-	-	-	11	-	78	-	5	2	-	3	2
La.	5	-	-	-	-	-	30	5	164	2	9	10	-	-	-
Okla.	6	-	8	-	-	2	8	34	150	-	24	31	-	1	-
Tex.	16	-	1	-	-	176	46	6	106	-	27	-	-	3	3
MOUNTAIN	13	-	116	-	2	400	42	7	133	22	322	72	2	6	16
Mont.	1	-	-	-	-	91	-	-	2	-	1	3	-	-	-
Idaho	-	-	-	-	1	-	4	-	1	5	242	27	-	-	1
Wyo.	-	-	-	-	-	2	-	-	2	-	1	2	-	-	1
Colo.	7	-	116	-	1	5	10	-	25	4	13	17	-	2	-
N. Mex.	1	-	-	-	-	297	10	N	N	-	1	4	-	-	-
Ariz.	2	-	-	-	-	4	10	6	90	13	44	18	-	-	4
Utah	1	-	-	-	-	-	7	-	3	-	19	1	2	3	10
Nev.	1	-	-	-	-	1	1	1	10	-	1	-	-	1	-
PACIFIC	105	4	246	-	32	552	457	13	288	9	152	205	3	50	109
Wash.	8	1	2	-	-	1	39	2	16	7	39	28	-	-	-
Oreg.	6	-	1	-	-	35	23	N	N	-	4	14	-	-	1
Calif.	88	3	241	-	29	512	377	8	259	2	87	81	3	42	75
Alaska	2	-	-	-	-	-	5	1	6	-	3	3	-	-	-
Hawaii	3	-	1	-	3	4	13	2	7	-	19	79	-	8	33
Guam	-	-	-	-	1	2	-	-	2	-	-	-	-	1	1
P.R.	1	12	171	-	-	407	6	-	5	-	6	12	-	1	2
V.I.	-	-	-	-	-	-	-	1	12	-	-	-	-	-	-
Amer. Samoa	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
C.N.M.I.	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-

*For measles only, imported cases includes both out-of-state and international importations.
N: Not notifiable U: Unavailable †International §Out-of-state

TABLE III. (Cont'd.) Cases of specified notifiable diseases, United States, weeks ending June 4, 1988 and June 6, 1987 (22nd Week)

Reporting Area	Syphilis (Civilian) (Primary & Secondary)		Toxic-shock Syndrome	Tuberculosis		Tula-remia	Typhoid Fever	Typhus Fever (Tick-borne) (RMSF)	Rabies, Animal
	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1987	Cum. 1988	Cum. 1988	Cum. 1988	Cum. 1988
UNITED STATES	15,850	14,217	121	8,101	8,507	50	142	82	1,682
NEW ENGLAND	404	221	11	159	271	1	11	1	3
Maine	5	1	2	3	15	-	-	-	1
N.H.	4	2	3	-	5	-	-	-	2
Vt.	1	1	2	1	6	-	1	-	-
Mass.	173	108	4	96	143	1	7	1	-
R.I.	13	6	-	11	24	-	-	-	-
Conn.	208	103	-	48	78	-	3	-	-
MID. ATLANTIC	3,296	2,586	19	1,457	1,489	-	22	2	174
Upstate N.Y.	213	92	9	231	237	-	4	1	1
N.Y. City	2,113	1,841	2	668	720	-	8	1	-
N.J.	367	274	3	268	261	-	10	-	-
Pa.	603	379	5	290	271	-	-	-	173
E.N. CENTRAL	483	419	19	923	980	1	15	6	45
Ohio	50	48	15	169	191	-	4	6	-
Ind.	21	27	-	91	101	-	2	-	13
Ill.	240	233	-	380	397	-	6	-	8
Mich.	157	76	4	231	252	1	2	-	5
Wis.	15	34	-	52	39	-	1	-	19
W.N. CENTRAL	97	60	15	213	248	22	4	11	207
Minn.	8	6	1	38	62	-	2	-	76
Iowa	10	11	3	16	10	-	-	-	13
Mo.	54	27	6	104	136	16	2	8	5
N. Dak.	1	-	-	3	4	-	-	-	42
S. Dak.	5	5	1	19	9	3	-	1	54
Nebr.	13	7	2	7	11	2	-	-	6
Kans.	6	4	2	26	16	1	-	2	11
S. ATLANTIC	5,634	4,846	10	1,774	1,727	4	17	25	564
Del.	57	38	1	17	18	1	-	-	19
Md.	305	253	1	184	143	-	1	4	149
D.C.	256	148	-	80	57	-	-	-	4
Va.	184	112	-	186	170	2	7	3	182
W. Va.	5	5	-	34	50	-	-	1	44
N.C.	322	263	5	140	173	-	1	13	-
S.C.	251	319	-	182	153	-	-	3	31
Ga.	916	670	-	288	271	1	2	1	98
Fla.	3,338	3,038	3	665	692	-	6	-	37
E.S. CENTRAL	862	828	12	655	729	4	2	11	123
Ky.	28	6	5	177	187	3	1	1	58
Tenn.	364	360	4	193	219	-	-	7	32
Ala.	248	204	3	204	229	-	1	3	33
Miss.	212	258	-	81	94	1	-	-	-
W.S. CENTRAL	1,751	1,788	12	1,027	948	11	6	21	253
Ark.	98	88	-	101	104	5	-	1	41
La.	340	309	-	150	104	-	2	-	-
Okla.	72	76	4	94	94	6	-	17	19
Tex.	1,241	1,315	8	682	646	-	4	3	193
MOUNTAIN	299	308	13	182	243	5	6	4	147
Mont.	2	8	-	5	8	-	1	3	108
Idaho	-	3	2	2	16	-	-	1	-
Wyo.	1	1	-	1	1	-	-	-	15
Colo.	41	45	2	15	55	4	3	-	2
N. Mex.	22	29	-	38	39	1	1	-	4
Ariz.	77	146	5	97	108	-	1	-	17
Utah	9	12	4	-	6	-	-	-	1
Nev.	147	64	-	24	10	-	-	-	-
PACIFIC	3,034	3,162	10	1,711	1,872	2	59	1	166
Wash.	73	63	2	100	102	-	3	-	-
Oreg.	119	109	-	58	52	-	5	-	-
Calif.	2,817	2,982	8	1,467	1,602	-	49	1	160
Alaska	6	2	-	18	30	2	-	-	6
Hawaii	19	6	-	67	86	-	2	-	-
Guam	1	2	-	7	4	-	-	-	-
P.R.	287	428	-	91	117	-	2	-	31
V.I.	1	3	-	3	2	-	-	-	-
Amer. Samoa	-	2	-	-	-	-	-	-	-
C.N.M.I.	1	-	-	8	-	-	-	-	-

U: Unavailable

TABLE IV. Deaths in 121 U.S. cities,* week ending June 4, 1988 (22nd Week)

Reporting Area	All Causes, By Age (Years)						P&I**
	All Ages	≥65	45-64	25-44	1-24	<1	Total
NEW ENGLAND	628	438	122	36	15	17	42
Boston, Mass.	154	98	35	10	5	6	12
Bridgeport, Conn.	54	34	12	7	-	1	1
Cambridge, Mass.	24	18	3	2	1	-	2
Fall River, Mass.	44	35	6	2	1	-	4
Hartford, Conn.	53	40	10	1	2	-	-
Lowell, Mass.	25	15	7	3	-	-	1
Lynn, Mass.	12	12	-	-	-	-	-
New Bedford, Mass.	34	19	10	2	2	1	-
New Haven, Conn.	49	34	7	3	2	3	5
Providence, R.I.	36	28	7	-	-	1	1
Somerville, Mass.	6	3	2	1	-	-	1
Springfield, Mass.	38	24	6	3	-	3	3
Waterbury, Conn.	41	32	7	1	1	-	5
Worcester, Mass.	60	46	10	1	1	2	7
MID. ATLANTIC	2,589	1,711	513	267	58	50	142
Albany, N.Y.	61	44	11	1	2	3	-
Allentown, Pa.	15	12	3	-	-	-	1
Buffalo, N.Y.	158	110	35	9	2	2	12
Camden, N.J.	27	15	8	3	-	1	1
Elizabeth, N.J.	23	11	9	3	-	-	1
Erie, Pa.†	38	32	7	-	-	-	3
Jersey City, N.J.	52	35	10	6	-	1	1
N.Y. City, N.Y.	1,372	890	280	161	33	28	66
Newark, N.J.	63	21	16	17	5	4	4
Paterson, N.J.	31	18	9	3	1	-	1
Philadelphia, Pa.	312	205	59	35	10	3	18
Pittsburgh, Pa.†	64	46	14	2	1	1	1
Reading, Pa.	26	21	3	1	-	1	1
Rochester, N.Y.	134	93	23	13	2	3	15
Schenectady, N.Y.	22	17	5	-	-	-	-
Scranton, Pa.†	34	27	6	-	-	1	3
Syracuse, N.Y.	82	54	20	6	-	2	6
Trenton, N.J.	39	23	9	6	1	-	1
Utica, N.Y.	27	22	4	1	-	-	4
Yonkers, N.Y.	18	15	2	-	1	-	1
E.N. CENTRAL	2,122	1,408	440	154	61	61	91
Akron, Ohio	58	45	6	2	2	3	1
Canton, Ohio	32	24	4	2	1	1	4
Chicago, Ill.§	564	362	125	45	10	22	16
Cincinnati, Ohio	106	72	26	5	2	1	10
Cleveland, Ohio	130	85	23	11	8	3	5
Columbus, Ohio	123	71	34	8	7	3	3
Dayton, Ohio	120	79	25	10	3	3	3
Detroit, Mich.	214	122	44	29	6	13	7
Evansville, Ind.	34	28	6	-	-	-	2
Fort Wayne, Ind.	45	29	11	-	3	2	-
Gary, Ind.	14	9	4	-	1	-	1
Grand Rapids, Mich.	54	32	14	5	1	2	4
Indianapolis, Ind.	157	94	46	12	4	1	2
Madison, Wis.	37	25	5	6	-	1	-
Milwaukee, Wis.	126	93	20	4	7	2	3
Peoria, Ill.	51	42	4	2	-	3	7
Rockford, Ill.	39	29	6	1	3	-	6
South Bend, Ind.	38	30	6	1	1	-	4
Toledo, Ohio	121	86	25	9	1	-	13
Youngstown, Ohio	59	49	6	2	1	1	-
W.N. CENTRAL	645	440	123	36	24	22	31
Des Moines, Iowa	33	25	6	-	2	-	2
Duluth, Minn.	28	25	2	1	-	-	2
Kansas City, Kans.	19	12	5	-	1	1	1
Kansas City, Mo.	120	75	30	7	5	3	3
Lincoln, Nebr.	21	17	4	-	-	-	2
Minneapolis, Minn.	144	92	29	11	6	6	6
Omaha, Nebr.	68	38	16	3	7	4	3
St. Louis, Mo.	110	80	17	7	2	4	2
St. Paul, Minn.	41	30	7	2	1	1	-
Wichita, Kans.	61	46	7	5	-	3	8
S. ATLANTIC	1,215	756	256	126	38	39	61
Atlanta, Ga.	155	92	38	16	7	2	6
Baltimore, Md.	285	154	61	29	11	10	13
Charlotte, N.C.	58	38	11	3	3	3	5
Jacksonville, Fla.	107	75	24	7	1	-	5
Miami, Fla.	104	59	19	20	4	2	-
Norfolk, Va.	52	34	10	3	3	2	3
Richmond, Va.	74	42	21	5	3	3	8
Savannah, Ga.	59	45	11	3	-	-	6
St. Petersburg, Fla.	63	50	8	1	2	2	2
Tampa, Fla.	55	38	8	7	-	2	7
Washington, D.C.	203	113	43	30	4	13	6
Wilmington, Del.	20	16	2	2	-	-	-
E.S. CENTRAL	645	448	125	31	19	22	46
Birmingham, Ala.	87	54	22	3	3	5	-
Chattanooga, Tenn.	36	28	5	2	-	1	6
Knoxville, Tenn.	67	51	12	1	1	2	6
Louisville, Ky.	80	49	23	3	2	3	1
Memphis, Tenn.	136	93	24	10	5	4	18
Mobile, Ala.	78	60	11	2	2	1	2
Montgomery, Ala.	43	30	7	1	3	2	6
Nashville, Tenn.	120	83	21	9	3	4	7
W.S. CENTRAL	1,128	722	245	87	40	34	36
Austin, Tex.	44	29	7	6	2	-	3
Baton Rouge, La.	33	23	5	2	3	-	2
Corpus Christi, Tex.§	40	31	9	-	-	-	1
Dallas, Tex.	137	81	28	16	7	5	-
El Paso, Tex.	51	31	13	2	2	3	2
Fort Worth, Tex	82	52	18	4	3	5	3
Houston, Tex.§	308	176	74	34	13	11	7
Little Rock, Ark.	52	31	14	2	-	5	3
New Orleans, La.	90	53	27	7	3	-	1
San Antonio, Tex.	174	127	29	8	7	3	7
Shreveport, La.	40	28	10	2	-	-	4
Tulsa, Okla.	77	60	11	4	-	2	3
MOUNTAIN	601	392	122	52	16	19	25
Albuquerque, N. Mex.	80	57	11	11	1	-	6
Colo. Springs, Colo.	39	25	8	4	1	1	1
Denver, Colo.	92	63	20	6	1	2	2
Las Vegas, Nev.	94	61	21	8	3	1	5
Ogden, Utah	26	19	5	2	-	-	3
Phoenix, Ariz.	105	57	26	8	4	10	2
Pueblo, Colo.	27	18	8	1	-	-	2
Salt Lake City, Utah	53	29	11	5	5	3	3
Tucson, Ariz.	85	63	12	7	1	2	2
PACIFIC	1,627	1,036	327	149	58	49	76
Berkeley, Calif.	12	10	1	1	-	-	1
Fresno, Calif.	100	69	17	8	2	4	11
Glendale, Calif.	24	20	2	1	-	1	2
Honolulu, Hawaii	54	35	13	5	-	1	8
Long Beach, Calif.	94	69	18	5	2	1	4
Los Angeles Calif.	312	181	73	36	15	7	7
Oakland, Calif.	44	27	12	2	2	1	1
Pasadena, Calif.§	33	27	4	1	-	1	2
Portland, Oreg.	121	83	18	13	5	2	4
Sacramento, Calif.	132	75	38	9	4	6	10
San Diego, Calif.	125	72	26	14	3	10	6
San Francisco, Calif.	147	82	28	26	4	7	4
San Jose, Calif.	179	112	38	11	10	7	10
Seattle, Wash.	156	106	27	12	8	3	3
Spokane, Wash.	50	39	5	3	-	3	2
Tacoma, Wash.	44	32	7	2	3	-	1
TOTAL	11,210††	7,351	2,273	938	329	313	550

*Mortality data in this table are voluntarily reported from 121 cities in the United states, most of which have populations of 100,000 or more. A death is reported by the place of its occurrence and by the week that the death certificate was filed. Fetal deaths are not included.

**Pneumonia and influenza.

†Because of changes in reporting methods in these 3 Pennsylvania cities, these numbers are partial counts for the current week. Complete counts will be available in 4 to 6 weeks.

††Total includes unknown ages.

§Data not available. Figures are estimates based on average of past available 4 weeks.

Hepatitis B Virus — Continued

14. Kane MA, Hadler SC, Margolis HS, Maynard JE. Routine prenatal screening for hepatitis B surface antigen. JAMA 1988;259:408–9.
15. Hershow RC, Hadler SC, Kane MA. Adoption of children from countries with endemic hepatitis B: transmission risks and medical issues. Pediatr Infect Dis 1987;6:431–7.
16. Beasley RP, Stevens CE. Vertical transmission of HBV and interruption with globulin. In: Vyas GN, Cohen SN, Schmid R, eds. Viral hepatitis: a contemporary assessment of etiology, epidemiology, pathogenesis and prevention. Philadelphia: Franklin Institute Press, 1978:333–45.
17. Sinatra FR, Shah P, Weissman JY, Thomas DW, Merritt RJ, Tong MJ. Perinatal transmitted acute icteric hepatitis B in infants born to hepatitis B surface antigen-positive and anti-hepatitis Be-positive carrier mothers. Pediatrics 1982;70:557–9.
18. Delaplane D, Yogev R, Crussi F, Shulman ST. Fatal hepatitis B in early infancy: the importance of identifying HBsAg-positive pregnant women and providing immunoprophylaxis to their newborns. Pediatrics 1983;72:176–80.
19. Immunization Practices Advisory Committee. Recommendations for protection against viral hepatitis. MMWR 1985;34:313–24,329–35.
20. Klontz KC. A program to provide hepatitis B immunoprophylaxis to infants born to HBsAg-positive Asian and Pacific Island women. West J Med 1987;146:195–9.
21. McMahon BJ, Rhoades ER, Heyward WL, et al. A comprehensive programme to reduce the incidence of hepatitis B virus infection and its sequelae in Alaskan Natives. Lancet 1987;2:1134–6.
22. Xu Z-Y, Liu C-B, Francis DP, et al. Prevention of perinatal acquisition of hepatitis B virus carriage using vaccine: preliminary report of a randomized, double-blind placebo-controlled and comparative trial. Pediatrics 1985;76:713–8.
23. Coursaget P, Yvonnet B, Chotard J, et al. Seven-year study of hepatitis B vaccine efficacy in infants from an endemic area (Senegal). Lancet 1986;2:1143–5.

Imported Human Rabies — Australia, 1987

In November 1987, the illness of a 10-year-old Australian boy who had died of acute encephalitis 4 months earlier was confirmed as being rabies. This is the first laboratory-confirmed case of human or animal rabies ever reported from Australia.

The child had traveled with his mother to India, Pakistan, Nepal, Singapore, and Thailand between February and October of 1986, but no animal bites were reported during this period. He remained well until June 23, 1987, when headache developed, followed by fever, vomiting, and chills. During the next few days, he became anorexic, had a few episodes of delirium at night, and had pain in his right arm. Eight days after the onset of illness, incoordination and diplopia developed, along with a progressive weakness in his legs. When admitted to a local hospital, the patient had palsies of the sixth cranial nerve on the right side and of the seventh cranial nerve bilaterally. Analysis of a cerebrospinal fluid specimen showed normal cell counts and normal protein and glucose levels. A diagnosis of atypical Guillain-Barré syndrome versus encephalitis was made, and on the 10th day of illness the patient was transferred to a regional medical center. At that time, he was unable to walk, and his reflexes were decreased on the right side. An electroencephalogram showed slow wave activity consistent with a diffuse encephalitis, and a computerized axial tomography scan was normal. A repeated lumbar puncture on the 12th day of illness showed 50 white blood cells/mm^3, 18 red blood cells/mm^3, elevated glucose, and normal protein levels. The patient was temporarily intubated because of irregular respiration. On the 14th day of illness, inappropriate antidiuretic hormone secretion, upper airway obstruction, and pneumonia developed, and the left lung collapsed. Seizures began 2 days later. The patient became comatose on the 19th day of illness, and he died 4 days later.

Human Rabies — Continued

Hospital pathologists found eosinophilic intracytoplasmic inclusions, suggestive of Negri bodies, on fixed sections of brain tissue. A serum sample taken on the 21st day of illness had a rabies neutralizing antibody titer of 1,400 when analyzed at a reference laboratory 4 months later. No specimens were available for virus isolation.

In December, extensive interviews with relatives, friends, and other contacts of the patient revealed that the patient, an animal lover, had been injured by two animals in the 2 years before his death. He was severely scratched by a neighbor's dog 2 months before his onset of illness, but the dog remained healthy and did not have rabies antibodies when tested in December 1987. However, according to a travel companion, the patient was bitten on a finger by a wild monkey at a marketplace in northern India 16 months before the onset of illness. This incident was not reported to the boy's mother. A photograph of the patient feeding the monkeys at this marketplace was found in a school project he had prepared.

Rabies postexposure prophylaxis was recommended for nine health-care workers and four family members and friends who may have been exposed to the patient's saliva or nerve tissue during his illness.

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Editorial Note: All available data indicate that this was an imported case of rabies. The only previous report of animal or human rabies in Australia is poorly documented, but, in 1867, a child and a dog from Tasmania had suspected rabies (*1*). Both animal and human rabies have been reported from all the countries in which the patient traveled except Singapore (*2*). (Nepal did not contribute to that survey.) The monkey bite in northern India 16 months before the onset of illness must be considered the probable exposure, but the patient might have received other unreported bites while traveling in Asia. If the patient was exposed in Asia, the incubation period would have been between 8 and 16 months. In one large study of human rabies, approximately 1% of cases had incubation periods of over 1 year (*3*). Cases of monkey-transmitted human rabies are rare; however, one extremely long incubation period (37.5 months) was reported (*4*).

In the Australian boy's case, paralysis dominated the clinical picture. The clinical picture and the prolonged course before the onset of coma are consistent with the paralytic form of rabies, which occurs in approximately 20% of cases (*3*).

Since Australia is considered a rabies-free country (*2*), animal rabies vaccination is not required and animals that bite people are not quarantined. If animal rabies should become endemic in Australia, an estimated 38,000 people per year might have to receive postexposure prophylaxis (*5*). In addition, millions of dollars would have to be spent on animal rabies vaccination and quarantine. Wild animals that might become reservoirs if rabies should be introduced into Australia include the dingo (a wild dog), red fox, feral cat, and bat. A limited serosurvey of bats in Queensland for rabies antibodies is in progress.

Human Rabies — Continued

This report emphasizes the importance of rabies preexposure prophylaxis for travelers visiting rabies-endemic countries for more than 30 days (*6*), especially children who are likely to have unrecognized or unreported exposures. Preexposure prophylaxis can be administered intramuscularly or intradermally (*7*); however, the intradermal regimen should be completed at least 30 days before departure and should not be used if the person is taking chloroquine for malaria chemoprophylaxis.

References

1. Bisseru B. Rabies. London: William Heinemann Medical Books, 1972:18.
2. World Health Organization. World survey of rabies XXII (for years 1984/85). Geneva: World Health Organization, Division of Communicable Diseases, 1987.
3. Hattwick MAW. Human rabies. Public Health Rev 1974;3:229–74.
4. Wilson JM, Hettiarachchi J, Wijesuriya LM. Presenting features and diagnosis of rabies. Lancet 1975;2:1139–40.
5. Nixon J, Pearn J, McGarn F. Dog bite injuries to children: potential rabies threat to Australia. Med J Aust 1980;1:175–6.
6. Centers for Disease Control. Health information for international travel 1987. Washington, DC: US Department of Health and Human Services, Public Health Service, 1987:108–12; DHHS publication no. (CDC)85-8280.
7. Immunization Practices Advisory Committee. Rabies prevention: supplementary statement on the preexposure use of human diploid cell rabies vaccine by the intradermal route. MMWR 1986;35:767–8.

Methemoglobinemia due to Occupational Exposure to Dinitrobenzene — Ohio, 1986

On April 23, 1986, five steam-press operators at an Ohio rubber plant became ill with symptoms including yellow discoloration of the hands, blue discoloration of the lips and nail beds, headache, nausea, chest pain, dizziness, confusion, and difficulty in concentrating. One worker suffered a seizure. Medical examinations showed that blood methemoglobin (MetHb) levels in the workers ranged from 3.8% to 41.2% (normal level ≤1%).

The workers had been using an adhesive to bond metal studs into rubber strips to be attached to automotive bumpers. When the outbreak occurred, officials of the company voluntarily stopped steam-press operations and asked that representatives from the Occupational Safety and Health Administration (OSHA) and the Ohio Industrial Commission investigate. Five days later, a plant supervisor operated the steam-press for about 2 hours so that an industrial hygienist with the Ohio Industrial Commission could take air samples. After the 2-hour simulation, the supervisor's blood MetHb level was 12.5%. Since the cause of the incident remained unknown 1 week later, plant management requested technical assistance from the National Institute for Occupational Safety and Health (NIOSH) (*1*).

The product being used is a solvent-borne adhesive that is composed of carbon black (<5% by weight), a proprietary curative system (<5% by weight), and xylene as a solvent (approximately 78% by weight). NIOSH personnel collected bulk samples from the lot ("old" lot) of adhesive used at the time of the outbreak and from a new lot that arrived after the outbreak. Samples were extracted with carbon disulfide and methanol, and the extracts were analyzed by using a gas chromatograph equipped with a flame ionization detector. Para-dinitrobenzene (p-DNB) was identified as a contaminant in the old lot of adhesive. So that concentrations of p-DNB in both the old and new lots could be determined, portions of the samples were extracted and

Methemoglobinemia — Continued

p-DNB standards were dissolved in acetone and analyzed by gas chromatography. The concentration of p-DNB in the old lot (1% by weight) was approximately 30 times that in the new lot (0.03% by weight).

The NIOSH investigation, in conjunction with that of the adhesive manufacturer, revealed that p-DNB had been inadvertently formed during the manufacture of one of the proprietary substances used as a base chemical in the adhesive. This p-DNB-contaminated chemical was then introduced into the adhesive during its formulation. When notified of these findings, the manufacturer of the adhesive recalled all lots thought to be contaminated with significant quantities of p-DNB. The manufacturer also revised the material safety data sheet for this adhesive to indicate that trace amounts of dinitrobenzene, which can cause cyanosis, may be present.

NIOSH recommended that workers in the plant use butyl rubber gloves to avoid skin contact with the dried adhesive and that plant management institute periodic medical monitoring of all workers exposed to the adhesive. After plant officials replaced the p-DNB-contaminated adhesive with another product and implemented the recommendations, the steam-press operations were resumed.

NIOSH personnel monitored workers throughout the first day of operation for any signs of p-DNB exposure. No workers complained of any symptoms during or after the work shift, and none showed evidence of cyanosis during physical examination. To monitor workers for MetHb, NIOSH also collected preshift and postshift blood samples from nine steam-press workers using the new adhesive and from six office workers (controls) with no chemical exposure. MetHb levels in all blood samples were within normal limits and remained essentially unchanged over the workday.

Reported by: Hazard Evaluations and Technical Assistance Br; Div of Surveillance, Hazard Evaluations, and Field Studies; National Institute for Occupational Safety and Health, CDC.

Editorial Note: Aromatic nitro compounds, such as p-DNB, are used in many industries, including the manufacture of dyes, explosives, pigments, insecticides, textiles, plastics, resins, elastomers, photographic developers, pharmaceuticals, plant-growth regulators, fuel additives, rubber accelerators, and antioxidants (*2,3*). Because of this wide variety of uses, the potential for occupational exposure to these compounds is great.

The present incident illustrates that excessive exposure to aromatic nitro compounds may cause adverse health effects. p-DNB is readily absorbed by the skin and exerts its adverse health effects via the formation of MetHb from hemoglobin (Hb). Accumulations of MetHb greater than 1% of the total Hb substantially reduce the blood's capacity to carry oxygen to tissues of the body. Symptoms of illness are generally related to the percentage of MetHb in the blood: cyanosis and headache occur first (in persons with greater than 15% MetHb); dizziness and fatigue appear next (with greater than 40% MetHb); and ataxia, shortness of breath, tachycardia, nausea, vomiting, and drowsiness follow and can progress to stupor, coma, and possibly death (when levels exceed 70% MetHb).

The overall effect of substances that form MetHb is known as the "cyanosis-anemia syndrome" (*4,5*). p-DNB ranks second among cyanosis-producing chemicals and is also potent in causing anemia (Table 1).

The current OSHA permissible exposure limit and the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value for p-DNB is 1 mg/m^3 based on an 8-hour time-weighted average, and the ACGIH notes that p-DNB can be absorbed cutaneously, which can contribute to overall exposure (*6*). Indeed,

Methemoglobinemia — Continued

the skin is the main route by which several fat-soluble aromatic nitro compounds, including p-DNB, enter the body. For this reason and also because many nitrobenzene derivatives have low vapor pressures and do not reach high levels in the air, measures of airborne concentrations alone may not be the best indicator of total exposure.

The incident at this plant demonstrates the adverse health effects of a common class of industrial chemicals—aromatic nitro compounds—and emphasizes that employers and employees should know the potential dangers of exposure to these substances as well as to toxic substances in general. Further, since the incident was caused by a contaminated base chemical, the prevention of future episodes also depends on careful quality control in manufacturing that chemical. The actions of company officials in stopping the steam-press operations and cooperating with NIOSH technical personnel led to the rapid and successful resolution of this problem.

TABLE 1. Ranking of 13 chemicals that form methemoglobin and potentially produce cyanosis and anemia*

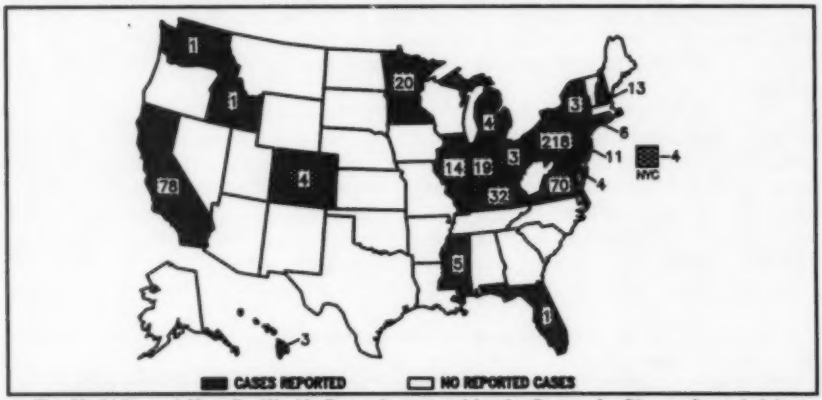
Chemical	Ranking for Production of	
	Cyanosis	Anemia
ortho-chloroaniline	1	—†
dinitrobenzene	2	5
meta-nitroaniline	3	—†
para-toluidine	4	4
nitrobenzene	5	1
meta-toluidine	6	12
ortho-nitrochlorobenzene	7	—†
aniline	8	10
para-dinitrosobenzene	9	11
ortho-toluidine	10	8
ortho-nitrotoluene	11	—†
nitronaphthalene	12	13
dichloroaniline	13	6

*Adapted from ranking of Linch (*5*); 1 = most potent, 13 = least potent.
†Insufficient data available for determining rank.

References

1. Stephenson RL, Gupta S, Rondinelli R. Health hazard evaluation report no. HETA 86-350-1815. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service, 1987.
2. Centers for Disease Control, Occupational Safety and Health Administration. NIOSH/OSHA occupational health guidelines for chemical hazards. Cincinnati, Ohio: US Department of Health and Human Services, Public Health Service; US Department of Labor, Occupational Safety and Health Administration, 1981; DHHS publication no. (NIOSH)81-123.
3. International Labour Office. Encyclopaedia of occupational health and safety. Vol 2. 3rd ed. Geneva: International Labour Organization, 1983:1355–6,1451–4.
4. Linch AL, Wuertz RL, Charsha RC. Chemical cyanosis and anemia control. In: Steere NV, ed. CRC handbook of laboratory safety. 2nd ed. West Palm Beach, Florida: CRC Press, 1978:342–78.
5. Linch AL. Biological monitoring for industrial exposure to cyanogenic aromatic nitro and amino compounds. Am Ind Hyg Assoc J 1974;35:426–32.
6. American Conference of Governmental Industrial Hygienists. Threshold limit values and biological exposure indices for 1987–1988. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists, 1987:20.

FIGURE I. Reported measles cases — United States, Weeks 18–21, 1988



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The data in this report are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. The editor welcomes accounts of interesting cases, outbreaks, environmental hazards, or other public health problems of current interest to health officials. Such reports and any other matters pertaining to editorial or other textual considerations should be addressed to: Editor, *Morbidity and Mortality Weekly Report*, Centers for Disease Control, Atlanta, Georgia 30333.